ARTHRITIS, OSTEOARTHRITIS AND RHEUMATOID ARTHRITIS

Arthritis refers to an inflammation of the joints and includes a group of more than 100 rheumatic diseases and other conditions, such as rheumatoid arthritis (RA), osteoarthritis (OA), psoriatic arthritis, gout, fibromyalgia and lupus. Although all these diseases can cause pain, stiffness and swelling in the joint, they have different etiologies and, in consequence, their therapies are quite different.

Any part of the body can become inflamed or painful from arthritis. Some rheumatic conditions can result in debilitating, even life-threatening complications or may affect other parts of the body including the muscles, bones and internal organs.

Arthritis can affect anyone at any age, its incidence increase with age, and, if left undiagnosed and untreated, many types of arthritis can cause irreversible damage to the joints, bones, organs, and skin.

OA is characterised pathologically by both focal loss of articular cartilage and marginal and central new bone formation. The etiology of OA is multifactorial and includes both generalised constitutional factors (for example, aging, sex, obesity, heredity, reproductive variables) and local adverse mechanical factors (for example, trauma, occupational and recreational usage, alignment) (1).

Specifically, the American College of Rheumatology (ACR) defines OA as a heterogeneous group of conditions that lead to joint symptoms and signs which are associated with defective integrity of articular cartilage, in addition to related changes in the underlying bone (sclerosis, subchondral bone collapse, bone cysts, and osteophyte formation) and at the joint margins (2).

OA is associated with pain (particularly use-related joint pain), stiffness and functional disability (3), but there is no strict correlation between joint symptoms and the extent or degree of pathological or radiographic changes.

Initially, OA is non-inflammatory and its onset is subtle and gradual, usually involving one or only few joints, being the joints more often affected knees, hip, hands and spine.

The objectives of the treatment of OA are alleviate pain, improve function, decrease disability and prevent or retard progression of the disease and its consequences (loss of articular cartilage and marginal and central new bone formation). Treatment of patients with OA should be individualized and tailored to the severity of the disease.

There are three classes of drugs for the treatment of OA: fast-acting drugs (analgesic and non-inflammatory agents - NSAIDs), slow-acting drugs (SYSADOA - Symptomatic slow-acting drugs for OA) that induce symptomatic relief, and disease modifying drugs (DMOAD - Disease modifying OA drugs) which have an effect on the progression of the pathological changes in osteoarthritis (4).

According to the European League Against Rheumatism (EULAR) recommendations for the treatment of knee osteoarthritis, paracetamol is the oral analgesic to try first and, when no responding to treatment, NSAIDs should be considered. When patient has an increased gastrointestinal risk, non-selective NSAIDs should be used together with effective gastroprotective agents, or other option should be selective COX-2 inhibitors. Topical applications with NSAID or capsaicin, are also commonly used. A useful alternative in patients in whom NSAIDs, including COX-2 selective inhibitors, are contraindicated, ineffective, and/or poorly tolerated are opioid analgesics, with or without paracetamol. SYSADOA (including glucosamine, chondroitin sulphate, diacerhein and hyaluronic acid) are featured with a delayed onset of action but its effect persists for some weeks after treatment discontinuation. Furthermore, SYSADOA may modify joint structure, acting as DMOAD drugs and they can be used alone or together

with NSAIDs. Intra-articular injection of long acting corticosteroid is indicated for flare of knee pain, especially if accompanied by effusion (1).

RA is an autoimmune disorder of unknown etiology characterized by symmetric, erosive synovitis and, in some cases, extraarticular involvement (5). Most patients experience a chronic fluctuating course of disease that, despite therapy, may result in progressive joint destruction, deformity, disability, and even premature death (6).

Features of the disease, that are amenable to improvement by existing pharmaceutical means, comprise pain, inflammation, physical disability and destruction of joints. The aims of the treatment of RA are relieve pain, decrease inflammatory synovitis, improve or sustain physical function and prevent structural joint damage (7).

There are three general classes of drug commonly used in the treatment of RA: NSAIDs, corticosterioids, and remittive agents or disease modifying anti-rheumatic drugs (DMARDs). NSAIDs and corticosteroids have a short onset of action while DMARDs can take several weeks or months to demonstrate a clinical effect. DMARDs include methotrexate, leflunomide, etanercept, infliximab, adalimumab, anakinra, antimalarials, gold salts, sulfasalazine, d-penicillamine, cyclosporin A, cyclophosphamide and azathioprine.

According to the EULAR recommendations for the management of early RA (8), patients at risk of developing persistent and/or erosive arthritis should be started with DMARDs as early as possible even if they do not fulfill established classification criteria for inflammatory rheumatological diseases yet, NSAIDs have to be considered in symptomatic patients after evaluation of gastrointestinal, renal and cardiovascular status. Systemic glucocorticoids reduce pain and swelling and should be considered as a (mainly temporary) adjunct therapy as part of the DMARD strategy. Intra-articular glucocorticoid injections should be considered for the relief of local symptoms of inflammation. Among the DMARDs, methotrexate is considered the anchor drug and should be used first in patients at risk of developing persistent disease.

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